These changes in reaction have already been discussed and referred to glycerin as the cause. Further investigations on this subject are now in progress.

SUMMARY.

The bacilli isolated from these three cases of presumable food infection do not correspond to the bovine type of bacilli in any one particular.

1. They were readily cultivated from nearly all the guinea-pigs inoculated, and they grew luxuriantly from the start on dog's serum. Subcultures on glycerin agar and glycerin bouillon grew vigorously after the first transfer from dog's serum.
2. Morphologically the bacilli were either fairly long—*i. e.*, they averaged about 2μ in length, or else they were very variable, polymorphic. The uniformly short, straight forms of the bovine type were absent.
3. In all cases the bacilli were of a very low order of virulence, lower even than that of many of the cultures of human origin studied heretofore.
4. None presented the reaction curve of true bovine bacilli.

We have, as yet, no satisfactory evidence concerning the degree of change, if any, which tubercle bacilli of bovine type may undergo in the human body. That changes could be as thoroughgoing as would be the case if we were inclined to attribute these cases of infection by way of the digestive tract to milk is at present quite inconceivable, and we must content ourselves by referring them to infection with bacilli of strictly human origin.

A STUDY OF CIRCUMSCRIBED AND DIFFUSE PULSATION OF THE WALL OF THE THORAX.

WITH THE REPORT OF A CASE OF PULSATING HÆMOTHORAX.

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Pulsation of the wall of the thorax is a physical sign varying so much in extent and degree that it is impossible to ascribe to it any general significance. Premising that it is invariably due to pulsation transmitted from the heart or large bloodvessels, with the exception of an apparent, not a real, pulsation due to rhythmical muscular contractions, the only classification that is possible is (1) pulsation without structural alteration in the walls of the thorax or in the tissues lying between the walls and the heart and bloodvessels, and (2) pulsations in which these structural changes exist and may be supposed to be the transmitting agents. In the former group are included all those forms of pulsation due to increase in the activity of the heart, where this increase is due to structural alteration or functional conditions, such as increased muscular activity or irritability of the nerve supply. It is hardly necessary to speak of the former group; pulsation of the wall of the chest is common in cases of marked hypertrophy of the heart. It may be limited to the precordial region, or cause a general systolic shock in a large area of the wall of the chest, perhaps extending along the upper part of the abdomen. The systolic movement is often wave-like in character, appearing to follow the heart's contraction from the base toward the apex, and this wave often appears to continue beyond the apex, moving toward the abdominal wall. Sometimes the impulse is more distinct to the eye than the hand. It may reach its maximum intensity in the region of the apex of the heart, and not infrequently is associated with slight bulging of the precordium. The impulse that occurs after violent exertion or excitement is usually characterized by a general systolic shock involving the whole of the thorax, and sometimes causing a movement of the trunk and head. Pulsation, either localized or diffuse, may occur in thoracic aneurysms. These varieties are sufficiently familiar to be passed without further comment.

Another form of pulsation occurs the nature of which is not quite so clear. The following case was observed at my clinic at the Polyclinic Hospital:

L. J., a Russian Jew, aged twenty-six years. The heart was normal and not beating vigorously. There was distinct suprasternal pulsation; the abdominal aorta pulsated palpably and visibly; there was distinct negative pulsation in the veins of the shoulder, which were distended, but no diastolic pulse was present in the veins at the base of the neck. Pulsation of the liver could not be determined, and the organ was not enlarged below the border of the ribs. There were no signs of arteriosclerosis. *There was a distinct, though slight, systolic pulsation of the whole right side of the thorax.* This was not perceptible upon palpation, probably because it was too diffuse. The left side did not pulsate. There were no signs of pleural effusion on the right side. The pulmonary sounds were normal, and the percussion note was not impaired. The patient had a slight degree of gastroptosis.

In a somewhat similar case recently reported by Edwards, there was vigorous expansile pulsation on the left side near the base, which led to the suspicion of aneurysm of the aorta. At the autopsy the thoracic organs were found to be entirely normal, but the bloodvessels were atheromatous. A case has also been observed by La Fleur.

In the case I report I am led, for lack of any more satisfactory explanation, to suppose that the pulsation of the thorax was similar to that observed in the abdomen, and was possibly due to the rather voluminous pulse not infrequently present in persons with long, narrow thoraces and ptoses of the organs.

Of the abnormal pulsations in the wall of the thorax, due to organic changes, perhaps the most common and important is the systolic retraction that may be associated with pericardial adhesions. The most pronounced form is that in which, in addition to the retraction of the apex, there is also retraction in the epigastrium and in the lower interspaces in the left posterior axillary line, known as Broadbent's sign. This sign is not uncommon. I have observed it in several cases, but upon only one of these could an autopsy be obtained. This case was as follows:

W. L., aged forty-two years, had had syphilis and rheumatism. He had known that he had heart trouble for five or six years. The area of cardiac dulness extended from the right parasternal line to the left anterior axillary line. There was a loud, harsh murmur at the apex, systolic in time, not heard at the base. There was pulsation in the veins of the neck, and a pulsating liver throughout the entire time of observation, which was three years. There was systolic retraction at the apex and epigastrium, and also in the eighth, ninth, tenth, and eleventh interspaces posteriorly. The latter was very distinct, and served on numerous occasions for the demonstration of Broadbent's sign.[1] A diagnosis was made of mitral and tricuspid regurgitation and obliterative pericarditis. At the autopsy the heart was found lying horizontally in the thorax, and extending from the right anterior axillary line to the left posterior axillary line. The left auricle was enormously dilated, being about as large as the rest of the heart. There was a buttonhole mitral valve, a greatly dilated tricuspid orifice, and obliterative pericarditis and pleuritis.

Next in importance are the cases in which effusion of air or liquid or both has taken place into the pleural cavity. This forms by far the most interesting group, and will, therefore, require more extended consideration. The condition has been known since the oft-quoted case of De Baillou, which was reported in 1640. It was evidently seen by others, and was fairly well known in the eighteenth century, if we may judge from the remarks in Le Roy's *Aphorisms*. The first case, however, that was ever reported with any degree of accuracy, according to Comby, is that of Bérard, which was published in 1810. Later, MacDonnell, independently and in ignorance of Bérard's work, published three interesting observations, and Stokes published one, reported most unsatisfactorily, and yet, on account of the extraordinary character of some of the symptoms, particularly noteworthy. A number of other cases were subsequently published, some complete, some merely mentioned incidentally in the course of the discussion of other conditions. Finally, in 1883, Comby, in addition to reporting three cases of his own, made a careful study of the literature and was able to collect twenty-seven cases all told. Four years later Keppler also reviewed the literature, reporting one case of his own, and collecting 38 cases. In 1888 Rummo reported 2 cases and reviewed the literature. In 1895 Comby published a small monograph in which he described 47 cases. In 1897 Fuchs reported an additional case, and was able to find 13 other cases not in Keppler's collection, bringing the number up to 52 cases. In the mean time, however, Wilson, of Philadelphia, in 1893, had reported 3 cases that had come under his observation, and had collected 65 cases from the literature, including those reported by Comby and Keppler. In 1900 Innocenti and Marsili reported 2 cases, and gave a careful review of the literature. Then, in 1903, Henry made a final report of his case, and added 15 cases not in Wilson's collection, making the total 80 cases. As far as I can discover, the total number of cases on record is 97, of which several are so incompletely reported that they are worthless for statistical purposes.

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[1] Broadbent's patient was a girl of nineteen who had been sick for two months. She was brought to the hospital two days before her death, with signs of pleural effusion on the left side. The heart was displaced to the right, but there was visible and palpable pulsation from the fifth interspace in the left nipple line to the xiphoid cartilage, and also in the tenth interspace from the spine to beyond the angle of the scapula. An aneurysm or malignant tumor was suspected, but at the autopsy obliterative pericarditis with adhesions of the pericardium to the central tendon of the diaphragm and the chest wall was found. Broadbent suggested that the pulsation posteriorly was due to the tugging of the heart on the diaphragm.

From a study of these cases we can recognize the following groups: (1) pulsating empyema and pulsating pyopneumothorax of necessity; (2) pulsating empyema and pulsating pyopneumothorax; (3) pulsating encapsulated empyema; (4) pulsating serous or hemorrhagic effusion; (5) pulsating extrapleural abscess.

The frequency of pulsations associated with various forms of pleural exudate is probably not great. Comby suspected that it was often overlooked, but after his excellent paper had called general attention to the subject the number of such cases reported was not very materially increased. Thus in Paris only four additional cases were observed in the period from 1882 to 1895. The condition may now be regarded as fairly well known. Several critical papers have been published in France by Comby; in Germany by Keppler and Fuchs; in America by Wilson and Henry; in Italy by Rummo and Innocenti and Marsili, and in England by West. Toft observed it in about 3 per cent. of his cases of empyema. Innocenti and Marsili estimate its frequency at 1 per cent. Many clinicians of large experience, as Millard, who have seen one case, and whose attention has been aroused, have not seen others, and other clinicians who have written about the subject, such as Rosenbach,[1] von Leube[1] and West have never observed any cases. It may be assumed, therefore, that such pulsations are uncommon, and must be produced by some unusual condition or group of conditions. Although these conditions must occur more or less independently of the nature of the pleural exudate, nevertheless the phenomena produced vary considerably in the different forms, and I believe it will be advisable to consider them separately. Before doing this it is well to discuss the different varieties of pulsation that may occur.

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[1] Diseases of the Bronchi, Lungs, and Pleura, edited by J. H. Musser, Philadelphia, 1902, p. 896.

It has been customary to divide the visible and palpable pulsations into those more or less diffuse in character and without tumor formation, and those in which a definite pulsating tumor occurs. Both may be present simultaneously in the same case. As the term "empyema of necessity" (pointing empyema) implies, tumors may appear in the wall of the thorax, and the pulsation may be limited strictly to them. It is, in fact, such tumors, often mistaken for aneurysm, that constituted the majority of the early reports of this condition, and gave rise to the belief, first expressed by Comby, that pulsation only occurs when the exudate is purulent. The pulsation in these tumors is systolic in time, that is to say, all observers, with the exception of MacDonnell and Stokes, state that it is synchronous with the contraction of the heart or with the pulse in the great vessels. MacDonnell states that in all his cases the pulsation was diastolic, but he does not say whether he compared it with the contraction of the heart or the pulsation of the radial artery, and it is improbable that in his cases it differed in character from that observed in the others. There is some question as to whether or not the pulsation is expansile. Ordinarily it is so described; but Duplay, who saw a case of Comby's in consultation, was able to demonstrate that it was not of the true expansile type observed in aneurysmal pulsation. Ordinarily the maximum pulsation is observed at the apex of the tumor, and not infrequently, when more than one tumor is present, one will pulsate and the other or others remain motionless. In some cases the pulsation does not develop *pari passu* with the tumor, but appears more or less suddenly sometime in its course, as in the case reported by Heyfelder. In other cases the pulsation is variously affected by different therapeutic measures that may be employed.

Rummo lays particular emphasis upon the difference between endopleural and exopleural pulsation. By the latter he means a pulsation that is visible and palpable externally, and by the former pulsation in a pleural exudate that can be detected only by instruments. For this purpose he used a hollow needle, a rubber tube, and a mercurial manometer, or, when the graphic method was employed, the tambour of a Marey sphygmograph. Then thrusting the needle into the pleural cavity he was able to get a tracing of the pulsation occurring in it. I have tried this three times, once in right-sided pleural effusion, and twice in left-sided pleural effusion, but have been unable to observe pulsation synchronous with the heart. I think it must be obvious that the distinction between the endopleural and exopleural pulsations is one rather of degree than of kind. If the endopleural pulsation becomes slightly stronger it will become exopleural without any change in the causative conditions. The classification of West, therefore, appears to be more satisfactory. He recognizes three forms: first, a general systolic shock over the thoracic wall, occurring in large effusions with much pressure; very much the same, he says, as the pulsation which may be transmitted through an intrathoracic tumor or the solid lung in pneumonia; second, a pulsation felt usually in front, which is due to the propagation through the fluid of a wave, and to relaxation of the walls of the thorax; third, an expansile type of pulsation which resembles the fluctuation observed in abscesses, and to produce which the surrounding portions must be rigid. The objection to West's classification is, I think, that it depends upon a hypothetical etiology. Clinically, I believe that a more satisfactory classification is as follows:

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[1] Diagnose der inneren Krankheiten, Leipzig, 1902, Bd. i. p. 175.

First. The diffuse pulsations of the thoracic walls. Of these there are two forms: first, a perceptible and palpable or merely palpable pulsation felt in one part of the thoracic wall. This gives a sensation not unlike that communicated by the wave that travels through an ascitic accumulation of fluid, but ordinarily not quite as distinct. In a case of chronic tuberculous empyema that I had an opportunity of observing in Dr. Allyn's wards at the Philadelphia Hospital I was able to demonstrate this form of pulsation to my class. It was felt from the eighth to the eleventh interspaces in the neighborhood of the posterior axillary line on the left side. It was not a pronounced pulsation and was not visible. The second form is a more powerful pulsation affecting the whole wall of the chest, visible and palpable, and even perceptible subjectively to the patient. The most remarkable case of this kind is that reported by Stokes, one of the earliest cases of pulsating pleurisy to become generally known. In this case the pulsation was so violent that it not only kept the patient awake at night, but even perceptibly shook the bed, and this in spite of the fact that the cardiac action was not greatly exaggerated. One of Béclère's cases exhibited the same form of pulsation.

Second. The circumscribed pulsations with tumor formation. These may be subdivided into four classes: first, cases of empyema or pyopneumothorax, in which, as a result of a local area of weakness, there is a bulging of the wall of the thorax where the pulsation occurs; second, the cases in which there has been perforation of the pleura, the pus from the empyema passing through the intercostal muscles, and giving rise to a subcutaneous abscess communicating with the empyema by a fistula. In both these classes there may also be diffuse pulsation; third, localized bulging and pulsation as a result of encapsulation of fluid in the pleural space, whether the fluid is purulent or serous in character; fourth, the formation of an abscess entirely outside of the thoracic cavity, usually arising from the periosteum of the ribs or sternum, and ordinarily tubercular in nature. Such tumors are comparatively rare. I have already discussed and illustrated the non-pulsating form in a previous paper.[1] Unless perforation from without inward has occurred it is difficult to understand how the pulsation is produced.

Third. A true expansile pulsation of the whole thorax, of which the only certain case apparently is the one I record, that is probably due in part to factors other than the mere accumulation of fluid in the pleural cavity, the displacement of the viscera, and the altered tension of the walls.

Rather than give brief abstracts of all the reported cases—which has already been done satisfactorily in this country by Wilson and continued almost to date by Henry—it has seemed to me more useful to summarize the results in the form of statistics. In order, however, to avoid the imputation of including imaginary cases, most unjustly made of Wilson by Fuchs and Innocenti and Marsili, I shall give a list of the references of all the cases I have been able to collect, arranged chronologically. These are altogether ninety-seven. Two of these cases (Potain and West) are mentioned incidentally in the discussion, in one instance of pulsating empyema, in the other of thoracic aneurysm. No details whatever are given, and they are, therefore, not considered. It has been customary since Keppler's paper appeared to include the case of Geigel[2] in the group of pulsating empyemas. In his report, however, Geigel states that the tumor below the ribs on the right side gave a distinct sense of fluctuation, but says nothing of pulsation. The case, therefore, has been omitted.

Woillez[3] mentions cases observed by Koelpin and Govelle, but Comby has been unable to find the original records:

Baillou. Empyema of Necessity. Paris, 1640. Quoted by Comby.

Pelletan. Extrapleural Abscess. Clinique chirurgicale ou mémoires et observations de chirurgie clinique, Paris, 1810, tome iii. p. 254.

Bérard. Empyema of Necessity. Du diagnostic dans les maladies chirurgicales de ses sources et de ses incertitudes et de ses erreures, Paris, 1836.

MacDonnell 1. Pyopneumothorax with Tumor. 2. Empyema of Necessity. 3. Empyema of Necessity. Dublin Journal of Medical Sciences, 1844, p. 1.

Wintrich. 1. Empyema of Necessity. 2. Empyema of Necessity. Virchow's Handbuch der speciellen Pathologie u. Therapie, 1854, Bd. v. pp. 259-301.

Stokes. Empyema. Diseases of the Heart and Aorta, Dublin, 1854, p. 607; Philadelphia, p. 623.

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[1] Philadelphia Medical Journal, April 5, 1902.

[2] Wurzburger med. Zeitschr., 1859, Bd. i.

[3] Traité clinique des maladies aiguës des organes respiratoires, Paris, 1872, p. 403.

Vidal. Mediastinal Abscess. Bull. de la Société anatomique, 1854, 29th year, p. 243.

Cruveilhier. Serous Effusion. Bull. de la Société anatomique, 1854, p. 244.

Asson. Empyema of Necessity. Giornale veneti di Scienze med., September, 1855.

Flint. Serous Effusion. Physical Exploration and Diagnosis of Diseases Affecting the Respiratory Organs, Philadelphia, 1856, p. 581.

Heyfelder. 1. Empyema of Necessity. 2. Circumscribed Pleural Abscess. Zeit. f. praktische Heilkunde, November, 1858, p. 860.

Aran. Empyema of Necessity. Gazette des hôpitaux, November, 1858, No. 108, p. 91.

Rees. Empyema of Necessity. British Medical Journal, August 21, 1858.

Wilks. Empyema of Necessity. Lancet, November 12, 1859, p. 481.

Baly. Serous Effusion. Lancet, November 12, 1859, p. 482.

Wiltshire (Chandler). Empyema of Necessity. Lancet, May 31, 1862, p. 573.

Chambers (Ash). Empyema of Necessity. Lancet, May 31, 1862, p. 573.

Lesage. Circumscribed Empyèma (Empyème pulsatil à répétition). Gazette des hôpitaux, October 31, 1865, p. 511.

Chvostek. 1. Empyema of Necessity. 2. Empyema of Necessity. 3. Empyema of Necessity. Wiener med. Blätte, 1865, p. 115.

Reyher. Periosteal Abscess. Berl. klin. Woch., 1868, p. 426.

Courbon (Le Double). Empyema of Necessity. Gazette des hôpitaux, May 24, 1870, p. 237.

Toft. 1. Empyema of Necessity. 2. Empyema of Necessity. 3. Empyema. Nord. med. Arkiv, 1871, Bd. iii., No. 7.

Walshe.[1] 1. Empyema. 2. Empyema. Practical Treatise of Diseases of the Lungs and Heart, 4th edition, 1871, p. 296.

Kemp. Empyema. Quoted by Walshe.

Traube. 1. Doubtful. 2. Empyema. Berl. klin. Woch., 1872, No. 7.

Shattuck. Empyema. International Clinics, January, 1872, p. 87.

Plagge. Empyema of Necessity. Beiträge z. Kenntniss d. pulsirenden Thoraxgeswulste. Memorabilien, 1872, No. 11, p. 481.

Müller. Empyema of Necessity. Berl. klin. Woch., January 22, 1872, p. 37.

Fraentzel. Empyema. Ziemssen's Handbuch, 1874-75, Bd. iv. 2d half, p. 301.

Lorenzutti. Empyema of Necessity. Annali universali di medicina e di chirurgia. Milano, 1875, vol. ccxxxii. p. 385.

Topham. Intrathoracic Abscess. Lancet, 1878, p. 756.

Dunin. Doubtful. Gazetta Lekarska. Quoted by Comby.

Brünniche. Extrapleural Abscess Communicating with Empyema. Hospitalstidende, 2 R., Bd. ix. p. 65; Virchow-Hirsch Jahresberichte, II., 1882, Bd. x. p. 146.

Comby. 1. Empyema. 2. Empyema. 3. Empyema. 4. Empyème (case of Duguet). Arch. générale de médecine, 1882, p. 549; l'empyème pulsatile, Paris, 1895.

Yeo. Empyema of Necessity. Lancet, February 23, 1884, p. 331.

Dillingham. Empyema. New York Medical Record, March 8, 1884.

Féréol. Pyopneumothorax. Proc. de l'acad. de médecine, 1884, No. 7, p. 155.

Broadbent. Serous effusion. Lancet, May 31, 1884, p. 971.

Pel. Circumscribed Empyema. Berl. klin. Woch., February 25, 1884, p. 113.

Ross. Circumscribed Empyema. Canadian Medical and Surgical Journal, May, 1885, p. 606.

Henry. Pyopneumothorax with Tumor. Proceedings of the Philadelphia County Medical Society, 1885, vol. iii.; New York Medical Journal, May, 1903.

Florand. Empyema. La France médicale, 1886, p. 158.

Codivilli. Empyema of Necessity. Thesis, Bologna, 1886, No. 97. Quoted by Rummo.

Cammareri. Empyema of Necessity. Gazetta degli Ospedali, 1886, No. 97, p. 772.

McCosh. Empyema of Necessity. Medical Record, January 30, 1887, p. 156.

Keppler. Circumscribed Pleural Effusion. Deutsche Archiv f. klinische Medicin, 1887, p. 220.

Borgiotti. Pyopneumothorax. Clin. med. in Bollet. de Soc. Cult. d. Scienzi Siena, 1886-87, p. 91.

Rummo. 1. Empyema. 2. Empyema. Riforma Medica, 1888, p. 1304.

Martinez. Seropneumothorax. Quoted by Rummo.

Millard. Empyema of Necessity. Gazette hebdom. d. méd. et de chirur., 7-12, 1899, p. 447.

Osler. Serous Effusion. THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, January 1889, p. 43.

Martin. Pyopneumothorax with Tumor. Revue méd. de la Suisse Romande, 1889, No. 2.

Bimmerman. Empyema. Geneeskundig Tydschrift voor Nederlandsch Indië, 1889, Bd. xviii. Quoted by Henry.

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[1] Walshe states that he has seen six cases altogether.

Monari. Empyema of Necessity. Gaz. degli Ospedali, August 6, 1890, No. 63, p. 498

Light. 1. Empyema of Necessity. 2. Empyema of Necessity. 3. Empyema of Necessity. Lancet, September 26, 1891, vol. ii. p. 704.

Wilson. 1. Pyopneumothorax with Tumor. 2. Empyema. 3. Pyopneumothorax. Transactions of the Association of American Physicians, Philadelphia, 1893, vol. viii.: Medical News, July 15, 1893.

Pittorelli. Empyema. Gaz. degli Ospedali, 1893, vol. xiv. p. 1260.

Watkins. Pyopneumothorax. Lancet, January 21, 1893, p. 133.

Osler. Pyopneumothorax. Transactions of the Association of American Physicians, 1893, p. 210. Discussion of Wilson's paper.

Potain. Extrapleural Abscess. Médecine moderne, November 29, 1893.

Béclère. 1. (Marieux) Empyema. 2. (Béclère's) Empyema. Bull. et mém. de la Société médicale des hôpitaux de Paris, 1894, tome xi. p. 339; tome xii. p 42.

Proust. Empyema. Ibid., 1894, p. 339.

James. Empyema of Necessity. International Clinics, 1895, vol. ii. p. 5.

Toulmin. Empyema of Necessity. University Medical Magazine, 1895, vol. vii. p. 853.

Roux. Empyema. La Province médicale, Lyon, 1896, tome x. p. 100.

Lepine. Serous Effusion. Lyon médicale, 1896, No. 8, tome lxxxi.; Centralbl. f. innere Med., 1897, p. 765.

Fuchs. Pyopneumothorax. Zeitschr. f. klinische Medicin, 1897, 32 Suppl. Hft., p. 255.

Sallés. 1. Pyopneumothorax. 2. Pyopneumothorax. La Province médicale, 1897, tome xi. p. 337. Quoted by Henry.

Cavazzani. Extrapleural Abscess. Gaz. degli Ospedali, 1897, vol. xii. p. 781.

West. Extrapleural Abscess. Medical Chirurgical Transactions, 1897, vol. lxxx. p. 239.

Innocenti and Marsili. 1. Empyema. 2. Hæmopyothorax. Riforma Medica, 1900, No. 261, p. 423.

Auerbach. Serous Effusion. Münchener med. Wochs., 1901, p. 1228.

McPhedran. Serohemorrhagic Effusion. Transactions of the Association of American Physicians, 1903, p. 334.

Kullman. Serous Effusion. Deut. med. Woch., 1904, p. 339.

Stanton. Empyema of Necessity. Unpublished. Read before the Medical Section of the College of Physicians of Philadelphia, 1904.

Sailer. Hæmothorax.

The attempt to classify the cases is only partially successful, but the following list appears to be approximately correct:

Cases of	empyema of necessity, that is, with tumor formation	34
"	pyopneumothorax with tumor formation	4
"	empyema without tumor formation	25
"	pyopneumothorax without tumor formation	8
"	serous or hemorrhagic effusion	12
"	circumscribed empyema	5
"	extrapleural abscess	5
"	uncertain nature	2

Properly speaking, then, we have of the cases with pus in the pleural cavity, 38 with tumor and 33 without tumor. There were 13 cases of intrapleural or extrapleural abscess, and the remaining cases were either non-purulent or their nature not definitely known. It is not at all impossible that some of the cases of supposed serous pleurisy may have been purulent. This is particularly true of the case reported by Flint, and it is certain that in one case (Keppler's) the fluid, which was clear at the time of the first puncture, subsequently became purulent.

Of the 95 cases, 63 were males 19 females, and in 13 the sex is not given. There are, therefore, nearly three and one-half times as many males as females. Twelve patients were under twelve years of age; 10 between the ages of ten and twenty; 32 between twenty and thirty; 14 between thirty and forty; 6 between forty and fifty; and 3 between fifty and sixty. In 18 the age is not given. Practically 90 per cent. of the cases occurred before the age of forty, and nearly 70 per cent. before the age of thirty. Pulsation of the wall of the thorax may therefore be regarded as distinctly a condition of early life. The youngest case (Light) was three years, the oldest (Cavazzani) was fifty-six years.

The only interest in the result of these cases is in determining whether pulsation increases the gravity of the prognosis or not. Of the 95 cases, 37 recovered, 27 died, 16 were improved (probably in many cases only temporarily), and in 20 the result is not stated, either because the report was made while the patient was still under observation, because the patient withdrew from observation, or because the case was mentioned incidentally with reference only to the pulsation. Therefore, the mortality of the cases whose termination is definitely known is 46.7 per cent. The following table shows the results for the different forms:

	Recovered.	Died.	Improved.	Not stated.
Empyema with tumor	11	10	6	7
Empyema without tumor	6	8	4	7
Pyopneumothorax with tumor	1	3	0	0
Pyopneumothorax without tumor	2	1	4	1
Serous effusion	4	3	2	3
Circumscribed empyema	3	1	1	0
Extrapleural abscess	3	2	0	0

It is hardly worth while to note the manner in which the cases were treated. Operations were performed upon 69 cases, and in 6 of these spontaneous rupture also occurred. Spontaneous rupture into the lung occurred three times, into the œsophagus once, and externally six times. No treatment was employed in 6 cases, and in 11 the reports do not furnish any information. The operations included practically all the operations for empyema: aspiration, drainage, or resection of one or more ribs. In the case of Bérard the patient punctured the supposed aneurysm with a needle. Topham's patient was kept in bed for three years before spontaneous rupture occurred. The majority of cases that recovered had persistent deformity, but in a few cases the thorax resumed its normal shape.

The position of the heart is of some interest. It is stated that it was displaced to the right in 53 cases, in all of which the lesion was on the left side. In 31 left-sided cases its position is not mentioned, but often it is obvious that it must have been displaced. In 3 right-sided cases its position was not mentioned. In 1 right-sided case (Borgiotti) it was displaced to the left. The degree of displacement varied considerably. In the case of Wiltshire it was very slightly displaced, but in many of the other cases the apex beat was in the right nipple line, or even beyond that point. It is definitely stated that it was not displaced in 9 cases (Stokes, Vidal, Reyher, Béclère, Light, Cavazzani, Innocenti and Marsili, Sailer, and McPhedran). Two of these (Vidal and Reyher) were extrapleural abscesses; one (Cavazzani) was apparently a circumscribed empyema. In one (McPhedran) exopericardial adhesions were suspected, but not demonstrated, and in a very remarkable case (Béclère) there was a funnel-shaped thorax extending 3½ cm. inward which prevented the displacement of the heart. For 3 cases (Stokes, Light, and Innocenti and Marsili) no reasons are given, and in the eighth case (Sailer) exopericardial and endopericardial adhesions were found at the autopsy. In the 3 cases reported by Toft, hinderances to the dislocation of the heart, in the form of adhesions or effusion on the right side, existed.

The causes assigned are various, and probably often incorrect. Tuberculosis—in all probability the commonest cause—is mentioned only eleven times, and in only two instances, apparently, confirmed in inoculations (Rummo, Béclère). Acute pleuritis of undetermined nature preceded the condition fifteen times. Chronic pleurisy occurred twice. Pneumonia of some form, with pleurisy or abscess, occurred in 13 cases. There were 4 cases in which an acute illness preceded the empyema. There was 1 case of pertussis. Pleural effusion is said to have been present once. Pneumothorax had apparently been present in one case before the empyema, but had disappeared before the pulsation was observed. Severe injury to the side occurred in 5 cases, exposure twice. In one remarkable instance (Fuchs) there was an attempt at abortion, followed by salpingitis, subdiaphragmatic abscess rupturing through the diaphragm and empyema. A few special cases cannot be grouped. In the case of Courbon there had been dyspnœa and palpitation for four years, with boring pain in the lumbar region. In a case of Innocenti and Marsili, which developed in a man aged forty-three years, there had been pleurisy, with a large effusion at the age of twenty-five. In the case of Martinez carcinoma was present. For some other cases there are merely indefinite statements, such as "pain in the side," "cough," which may indicate an undiscovered pleurisy or pleural effusion, or perhaps pulmonary tuberculosis, and for the rest no etiological factor is mentioned.

The most important part of our knowledge of these conditions is obtained from the records of the autopsies. I will, therefore, give brief abstracts of the post-mortem findings in the 21 cases in which autopsy was performed.

1. MacDonnell: The left lung was collapsed and bound firmly to the chest wall by adhesions; it was tuberculous. The pericardium was adherent to the right lung.

2. MacDonnell: The lungs were tuberculous and contained a large abscess cavity. The fifth and sixth ribs were eroded.

3. Vidal: There was pus in both pleural cavities, and a small pocket back of the sternum, the same size as the tumor and com-

municating with it through a hole. Both these pockets contained thick, creamy pus, and neither communicated with the pleura.

4. Heyfelder: Caries of the second, third, seventh, and eighth ribs; rupture of the pleura, and external empyema. The liver was displaced downward. There was no trace of tuberculosis.

5. Rees: The left side of the chest was retracted; the left lung was compressed and tuberculous; there was also tuberculous meningitis and pneumonia of the right lung.

6. Comby: Tuberculosis of both lungs; fetid gas and pus in the left pleura. The pericardium was adherent to both lungs; the mediastinum was displaced to the left, and the left lung collapsed.

7. Comby: Two litres of pus in the left pleura. The left lung was collapsed and adherent to the pericardium. It contained cheesy nodules. The pleura was not thickened.

8. Comby: The left pleural cavity contained about six litres of pus. The diaphragm was depressed and the mediastinum was displaced to the right. The surface of the pleura was covered with a false membrane. The left lung was solidified and adherent to the pericardium. The membrane covering it was perforated in two places. The right lung was tuberculous. The pericardium was not adherent.

9. Chvostek: The left thoracic cavity was filled with lymph; the left lung was compressed and displaced upward; there were recent pericardial adhesions; the pleura was ruptured in the fifth interspace at the site of the tumor.

10. Chvostek: The left clavicle was dislocated; several ribs were denuded and eroded, and the intercostal muscles were altered. The pleura was thickened and perforated at the site of the tumor; the pericardium was adherent to the tumor, and the lung compressed and solid.

11. Toft: A thick pseudomembrane enclosed the empyema, through which a perforation led to a subcutaneous abscess. The heart was attached to the chest wall. There was obliterative pericarditis and chronic peritonitis.

12. Toft: The left pleural cavity contained pus and air. A fistula communicated with the subcutaneous tumor. The heart was normal. Pus was present in the right pleural cavity.

13. Traube: There was massive purulent pleural and pericardial effusion. At the point of pulsation the pleura was destroyed. The pericardium was thickened; the lung compressed, and the upper lobe was atelectatic. The heart was displaced to the right.

14. Lorenzetti: The lung was adherent to the thoracic wall at several points. There was recent purulent pericarditis.

15. Dillingham: The left pleura contained some large blood clots whose source was not found; it was lined with pyogenic membrane. There was an adherent pericardium.

16. Broadbent: Obliterative pericarditis. The pericardium was adherent to the central tendon of the diaphragm and to the chest wall.

17. Pel: The heart was displaced slightly to the right. The pericardium was thickened and adherent. There were two encapsulated empyemas, one at the left border of the heart.

18. Cammareri: The parietal pleura was thickened; there was erosion of the ribs in several places; the lung was separated from the cavity by a false membrane; the left side of the mediastinum was thickened; and the left part of the thorax was larger than the right.

19. Light: There was left empyema; the left lung was collapsed; there was acute pleurisy of the right side and acute peritonitis. The heart was in its normal position.

20. Light: There was left empyema; extrapleural abscess communicating with the empyema by a fistula; the left pleura was thickened and adherent to the pericardium; the left lung was collapsed; the heart was twisted, and the pericardium contained eight ounces of clear fluid.

21. Fuchs: There was pyopneumothorax on the left side; the left lung was collapsed and partly adherent to the diaphragm; the heart and aorta were displaced to the right, and a subphrenic abscess communicated with the pleural cavity.

The most notable facts are: that the pericardium was involved in 8 cases, the left lung collapsed in 11 cases, the ribs were eroded in 3 cases, and the pleura perforated in 6 cases.

It is somewhat difficult to classify the cases into those in which pulsation is more or less diffuse without distinct evidence of tumor formation, and those in which tumors actually occur. In many cases the pulsating area is localized, although there may be no local bulging, and sometimes not sufficient distinction has been made between this form and circumscribed pulsation in a projection. As nearly as I have been able to determine there are 50 cases in which definite tumors have been recorded. Of these, 41 cases had a single tumor, 6 cases had 2 tumors, and 3 cases (Henry, Chvostek, and Toft) 3 tumors. Of these cases 37 were apparently empyemas of necessity, although some of them may have been encapsulated pleural abscesses; 11 were abscesses in some part of the thorax, either encapsulated empyemas or pleurisies, mediastinal abscesses, or extrapleural or periosteal abscesses, and 2 (Cruveilhier and Kuhlman) seem to have been cases of serous effusion with distinct local bulging or pointing. Altogether the locality of 62 tumors has been more or less definitely mentioned. Of these, 3 were situated on the right side and 58 on the left side. The 4 tumors situated on the right side were distributed as follows: in the second interspace at right border of sternum (Heyfelder); from second to fifth ribs, right border of sternum (Reyher); seventh interspace, right border of sternum (Wilks). Of those on the left side, 2 were not localized very accurately; were on the anterior aspect of the chest; in the axillary area, and 10 posteriorly. Of the 42 in the anterior aspect, one (Cruveilhier) had a conoid tumor presenting in front of the sternum; one (Vidal) a mediastinal abscess that had perforated the lower portion of the sternum just to the left; 2 (McCosh, Martin) below the left clavicle; 4 (Plagge, Muller, Light, Wilson) were rather extensive tumors of the left side of the chest, usually discolored and reducible; 19 occupied the various interspaces from the second to the eighth, often more than one, just to the left of the sternum. In one case (Toft) 3 tumors were found at different levels in this situation; 14 were found in the precordial region; 10 tumors in the posterior portion of the chest, of which 4 were below the angle of the scapula, and 4 between the crest of the ilium and the lower border of the ribs just to the left of the spinal column; 1 was in the tenth interspace to the left of the spinal column, and 1 was not distinctly located.

It appears from this analysis that the commonest situation is beneath the costal cartilages on either side of the sternum, 22 tumors having occurred in this situation. Fourteen tumors were in the precordial region; 4 beneath the angle of the scapula on the left side, and 4 wholly below the ribs in the left lumbar region. The other tumors were somewhat irregularly distributed, probably as a result of purely local conditions. The 2 tumors that occurred in association with serous effusion had unusual situations in front of the sternum and in the left hypochondrium.

Of the cases without tumor formation, 2 occurred on the right side (Keppler, Borgiotti). In Keppler's case there were 2 encapsulated pleural effusions which gave rise to diffuse pulsation of the lateral and anterior aspects of the chest. After these were evacuated the pulsation ceased in spite of the accumulation of pus later. In Borgiotti's case there was pyopneumothorax on the right side, and the whole of the right side of the chest was involved in the pulsation. Diffuse expansile pulsation was noted in 5 cases of empyema (Stokes, Béclère, Toft, Dillingham, and Rummel), and in 3 cases of pyopneumothorax (Féréol, Wilson, and Sallés). In 2 cases (Roux and Salles) the pulsation is said to have been diffuse, but its extent is not given. In 4 cases (Flint, Bimmerman, Pitorelli, and Proust) it is not distinctly localized. The pulsation is localized as follows: in the axillary region, 6 cases; in the posterior inferior aspect of the chest, 4 cases; in the anterior region either near the sternum, in the subclavicular, or in the mammary region, 6 cases. In the case of McPhedran pulsation was seen in the axilla if the patient lay upon her back, and in the back if she lay on the right side.

In 3 cases of serous pleurisy more or less localized pulsation was seen as follows: from the sternum to the left of the axillary line, and from the clavicle to the fourth rib (Osler); in the lower aspect of the left chest (Bailey) and in the left hypochondrium back of the false ribs (Lepine). The limited diffuse pulsation is seen in a much larger proportion of the cases in the axillary and lower posterior regions than is the circumscribed tumor.

Two of the cases (Martinez and Sailer) were hæmothorax. In the case of McPhedran the fluid on the first aspiration was bloody, but subsequently became clear. In the case of Dillingham there was apparently distinct empyema, but at the autopsy large blood clots were found in the pleural cavity. In the case of Innocenti and Marsili blood was also found in the pleural cavity upon aspiration. This patient had tuberculosis.

Martinez's patient was a man, aged twenty-one years, who had cough, pain in the left chest, and gradual progressive weakness. There was diffuse pulsation of the left side of the thorax, involving the epigastrium, more pronounced in the lower than in the upper portion, and a localized pulsation in the second, third, and fourth interspaces between the sternum and the left nipple line. There were all the usual signs of fluid in the left pleura, the heart was dislocated to the right, and its pulsations could be seen and felt in the second, third, and fourth interspaces between the right border of the sternum and the right axillary line. The spleen was low and quite palpable. An operation was performed, and 1500 c.c. of a serosanguinolent fluid was obtained. The heart returned to the left side, the pulsation ceased, and the patient appeared to have recovered.

Two months later he returned with the same symptoms; in a short time carcinoma of the lymph glands developed, and he died. The left thorax was 3½ cm. larger than the right.

The second case I observed at the Philadelphia Hospital. G. B., a West Indian negro, aged thirty years, and a laborer by occupation, was admitted, October 14, 1902. His family history was negative or unknown. He had rheumatism ten years ago; had indulged in alcohol moderately, and in tobacco to excess. For the past year he had suffered from dyspnœa on exertion, palpitation, and, for a time, cough. Three weeks before admission the cough had ceased, but he had suffered from intermittent sharp pain in or around the region of the heart. This became so severe that he was obliged to give up work, and finally entered the hospital. On the day of admission there was a loud systolic murmur heard over the heart, the whole chest, and transmitted loudly into the neck. There was a friction sound synchronous with the heart in the second interspace, just to the right of the sternum; the respiratory murmur over both lungs was normal. The following day the percussion note over the left thorax was found to be absolutely flat. Tactile fremitus and vocal resonance were absent; the breath sounds could not be heard. Over the whole of the left chest a very loud rasping systolic murmur was audible. There was distinct pulsation of the whole of the left side. On the following day (the 16th) I dictated the following notes:

The patient is slender and muscular; the skeleton is normal; the mucous membranes are pale. The pupils are normal and react to light; there is systolic expansile pulsation in the neck and in the suprasternal notch. There is heaving impulse over the upper portion of the left thorax, extending as far as the posterior axillary line. The maximum impulse is in the fifth interspace in the nipple line. There is distinct impulse in the sixth interspace one inch outside the nipple line. Pulsation can be seen and felt as far as the tenth interspace in the posterior axillary line, and is distinctly expansile in time. There is pulsation of the upper portion of the abdomen synchronous with the heart, and a vigorous downward pulsation in the left hypochondrium. There is systolic impulse in the neck visible in the suprasternal notch. Over the apex there is a faint systolic thrill and a diastolic shock. In the second interspace to the right of the sternum there is a faint systolic thrill and a diastolic shock. In the second interspace to the left there is a powerful heaving systolic impulse, a strong diastolic shock, and an almost imperceptible thrill. Toward the axilla the thrill becomes fainter, and the systolic impulse is very powerful. In the third and fourth interspaces the systolic and especially the diastolic impulses are strong and the thrill is very faint. The systolic impulse can be felt as far as the costal border, and merges into the powerful hypochondrial pulsation. There is no pulsation at the umbilicus. There is slight pulsation in the right hypochondrium from the median to the nipple line. The radial pulses are equal, small, and rhythmical. The liver dulness commences at the seventh rib and moves with the respiration. There is some dulness on the right side as far as the left border of the sternum. From the clavicle to the border of the ribs there is absolute flatness of the left side anteriorly. The whole left side of the chest is tender, especially in the neighborhood of the fourth and fifth ribs from the nipple to the axillary line. On the right side tactile fremitus is diminished, vocal resonance is lost, respirations are vesicular, expiration is faint. On the left side the breath sounds are entirely absent. Over both lungs anteriorly as well as posteriorly there is a harsh systolic murmur not accompanied by sounds, which is loudest over the left lung posteriorly, and faintest over the right lung posteriorly near the apex. In the sixth interspace the first sound is loud, the second is loud and booming, and there is a faint systolic murmur. From time to time a distinct splashing sound is also heard. In the fifth interspace the first sound is less distinct, there is a long, harsh, systolic murmur, and a moderately accentuated second sound. In the fourth interspace the first sound is fainter, the murmur is harsher, and the second sound more accentuated. In the third interspace the murmur is still harsher, the second sound somewhat fainter, and distinctly reduplicated. At the pulmonic cartilage a loud, harsh murmur almost drowns the other sounds. The second sound is distinctly reduplicated. On the right side of the sternum the systolic murmur is fainter, and the sounds are almost inaudible. The murmur becomes harsher toward the fifth interspace to the right. On the left side of the sternum the murmur is harsher above, but at the fifth interspace it is faint, and two loud sounds are heard. In the epigastrium a faint first and an accentuated second sound are heard. In the carotids there is a loud, harsh, systolic murmur. In the femoral arteries there are no notes, and only a faint systolic murmur is developed on pressure. *When the patient lies upon the right side the position of the right border of dulness is not changed.* The respirations are labored; expansion on the left side is diminished. The superficial lymph glands are not enlarged. The temperature is normal, and has been so since admission. The respirations vary between 20 and 30, the pulse averages 104. The urine shows traces of albumin, the specific gravity is 1010; there are no casts. The blood pressure with Oliver's tonometer is 130.

On October 17th, with the assistance of the resident physician, Dr. Holmes, I made an exploratory puncture and withdrew pure blood. Aspiration was therefore not performed. The condition of the patient now remained practically unchanged. He was evidently suffering; there was distinct expansile pulsation over the whole left thorax easily elicited by placing one hand in front and the other in the back. The pulsation in the left hypochondrium was about as forceful as the day before.

The diagnosis appeared somewhat obscure. The sudden development of the hæmothorax made a rupture of an aneurysm most likely, and this diagnosis was strengthened by the physical signs, especially the loud murmur heard all over the chest. It seemed difficult, however, to explain why there had been no displacement of the heart to the right. An *x*-ray picture was taken by Dr. G. E. Pfahler, which confirmed the physical diagnosis in so far as it showed dense blackness of the whole of the left side of the thorax, and no dislocation of the heart to the right. The patient died rather suddenly on the morning of October 18th, and at 2 P.M. of the same day Dr. Ellis performed the autopsy. The notes are as follows:

Autopsy. The body of a well-developed male negro. The external examination shows bulging of the left side of the chest that extends from the clavicle to a point about 2 cm. below the nipple, and from the left border of the sternum to the anterior axillary line. In the centre this area is raised approximately 2 cm. higher than the corresponding point on the opposite side. Reflexion of the superficial tissues shows that it is due to a prominence of the ribs and dilatation of the intercostal spaces. On the right side the diaphragm is at the lower border of the fifth rib; on the left it is convex downward, the lower portion reaching the lower border of the rib. There are old firm adhesions between the omentum, spleen, and diaphragm, and between the spleen and left lobe of the liver. The right pleural cavity is normal. The left contains about three litres of blood, about two-thirds of which is clotted, the clots being perfectly free in the cavity. Extending toward the left from a point corresponding to the left midclavicular line there is a fairly formed band of adhesions, 8 cm. long, between the pleura, covering the second and third ribs, and what is apparently a dilated portion of the aorta. The left lung is compressed backward and to the right; it is about the size of a fist, and entirely atelectatic. The long axis of the heart is more transverse than normal. The heart extends about 5 cm. to the left and 5 cm. to the right of the median line. The pericardium is almost universally adherent by fibrinous attachments which can be broken. Aside from a slight thickening of the mitral and tricuspid leaflets, the interior of the heart is normal. Three cm. from the aortic valves there is an opening 6 cm. in length in the posterior superior portion of the aorta which communicates with a sac which extends upward and to the left. The sac projects a distance of 12 cm. from the aorta; the transverse diameter is about 12 cm. At a distance of about 5 cm. from the aorta there is an irregular slit-like opening 1 cm. in length, and there is also a second opening 3 cm. from this ½ cm. in length. The interior of the sac contains several large areas of adherent laminated blood clots; the aorta contains a few atheromatous patches just below the valves. The organs of the abdomen show nothing of interest. The pathological diagnosis is ruptured aneurysm of the ascending and transverse portions of the arch of the aorta, atheroma of the aorta, atelectasis of the left lung, and left hæmothorax.

The actual mechanism by which these diffuse localized pulsations of the thoracic wall are produced has been the subject of some dispute. Unquestionably a number of factors are active, and confusion is due rather to the fact that stress has been laid upon different ones by different investigators than that there is much actual difference of opinion. The source of the pulsation must be the heart or the great vessels. The majority of writers consider the heart the source. The pulsation must be transmitted through the pericardium, the pleura, possibly through the lung, and through the fluid or air, or both, in the pleural cavity to the point at which it is manifested. For practical purposes the thorax may be considered to be a box closed at the sides and above by a bony framework which is somewhat more yielding on either side of the sternum and on either side of the spinal column near its lower border. Below it is closed by a muscular membrane which is always in a state of tension as a result of the elasticity of the lung. This box is divided by a vertical partition composed of two layers of the parietal pleura on the right and left sides, and between them the heart in its pericardial sac, the great vessels arising from its base, the trachea, the œsophagus, the nerves, minor bloodvessels, and glands of the mediastinal space. Under ordinary conditions the pulsations of the heart, with the exception of the apex, are entirely absorbed by the lungs, always in a state of negative pressure. It has been shown (Regnard) that with each pulsation of the heart the lungs contract slightly. If one lung is replaced by fluid under ordinary conditions pulsation will not be transmitted to the wall of the chest, because the pulsation of the heart is absorbed by the remaining lung. In the 3 cases in which I introduced a hollow needle communicating with a manometer, no cardiac pulsation could be observed; the respiratory movement, however, was in both cases considerable. In these cases there was sufficient positive pressure during inspiration to maintain the level of the fluid in a glass tube at the height of the shoulder, and during expiration it rose somewhat higher. In another case that I had examined carefully for palpable or visible pulsation, the moment the trocar was introduced the fluid spurted out with considerable force, indicating a high degree of positive pressure. I, therefore, doubt whether pulsation occurs in all cases of pleural effusion. Comby, who was the first to consider the question in a scientific manner, laid great stress upon the collapse of the lung. He believed that if the lung on the left side still contained air it was impossible for pulsation to be transmitted. In the cases that he analyzed his contention appeared to be supported, there being always actual evidence or reason to believe that the lung was completely collapsed. This, of course, is the usual condition in chronic empyema.

The theories which have been suggested may be classified as follows:

1. It is due chiefly to the violent action of the heart, the impulse being transmitted through the fluid exactly as the wave of fluctuation is transmitted through the fluid in ascites. This theory is held by MacDonnell, Fraentzel, and Wintrich. Le Roy and Aran, two of the earlier observers, seem to regard it as the most likely explanation, although their statements are not very clear. Traube supposes that in addition there must be a pericardial exudate. Keppler, Fuchs, Auerbach, Light, Bouveret, Innocenti and Marsili, and Henry consider violent action of the heart as an essential cause, but not the only one.

2. Those theories which depend upon the assumption of the existence of adhesions so limiting the fluid or bringing it into closer contact with the heart that the pulsations of the latter are more readily imparted to it. Keppler, for example, believes that the exudate must be in part encapsulated, then compressed by the thoracic wall, and brought into intimate association with the vigorously pulsating heart. Gueneau de Mussy and Broadbent lay especial stress upon pericardial adhesions. That these are sufficiently common in cases of pulsating pleural effusion, a considera-

tion of the autopsy records will demonstrate; but they do not appear to be invariably present, and therefore cannot, as Comby remarks, be the sole cause.

3. Several authors have discussed the possibility of a peculiar quality in the liquid. Comby, Toft, and others have supposed that only in cases of empyema did the pulsation exist. The preponderance of such cases lends considerable color to this view, but if the reports of serous and hemorrhagic effusion were not sufficient to disprove it, the ingenious experiment of McCosh, who filled the sac of an empyema with normal sterile salt solution after washing out the pus, and obtained a reproduction of the pulsation, would be sufficient to show that it had no basis in fact. Comby admits in his last publication that the character of the fluid is not of importance. Féréol's belief that there must be an accumulation of air under pressure has very little evidence to support it. In many cases a pneumothorax was not present. In others it certainly occurred after the puncture.

4. The observation that the withdrawal of a part of the fluid usually abolished the pulsation, and at the same time reduced the tension, led to the supposition that it was the intrathoracic tension, or, rather, the positive pressure in the liquid, that caused the pulsation.

This view has been particularly supported by Osler. It is also considered as one of the elements of explanation by Béclère and Bouveret, and meets with the approval of Toft, Henry, Lépine, Auerbach, Light, and, indeed, nearly all clinicians who have observed cases. Undoubtedly there must be a certain degree of pressure, otherwise the pulsation would not exist; but Lépine has observed that the pulsation may become more violent after the withdrawal of a portion of the fluid, or may even appear for the first time in a reaccumulation after one or more evacuations. In Toulmin's case the withdrawal of part of the liquid did not cause the pulsation to disappear, and he was led to doubt the importance of tension. Cases have also been observed in which more than one tumor was present, the pulsation occurring only in that which was most dependent, so that the mere degree of intrathoracic tension could not alone affect it. Walshe, who was one of the earliest to speculate upon this subject, believed that the tension had little to do with it, and that the pulsation was merely a manifestation of ability to transmit shocks normally found in all liquids.

5. A fifth group of explanations insist chiefly upon the fixation of the walls of the space that contains the pleural exudate. This, of course, comprises the diaphragm, mediastinum, and ribs. Destole believes that there must be immobilization of the diaphragm, partly as a result of the pressure of the pleural fluid which in time equalizes its contractile force and compresses it downward until it becomes comparable to an inert elastic membrane projecting into

the abdomen, partly by the formation of a tense false membrane upon the parietal layer of the pleura.

6. Akin to this, but expressed in a somewhat different manner, is the theory that the pulsation is manifested when the tension of the mediastinum and diaphragm becomes greater than that of the chest wall. Light gives the following explanation: the resistance of the diaphragm, mediastinum, and lung must be augmented until it becomes greater than that of the intercostal spaces. This may be produced either if the effusion is very great, or if there is a thick membrane lining the pleura. Bouveret regards the increase in the resistance of the diaphragm and mediastinum as essential to the occurrence of pulsation. Béclère believes that he has proven by experiments upon the cadaver by means of the Roentgen rays, and by means of observations upon a patient with a funnel-shaped chest, that unless the resistance of a fibrinous mediastinitis is greater than that of the intercostal spaces, pulsation is not manifest. Auerbach agrees with him, and suggests that the fact that pulsation of the heart is more vigorous on the left than on the right side explains why pulsation is more common in cases of left-sided pleurisies. In all of Toft's cases fixation of the mediastinum was favored by obstruction to the dislocation of the heart. Essentially the same idea is expressed by those who believe that the weakening of the resistance of the wall of the thorax is the most important factor. Strongly in favor of this view is, of course, the fact that pulsation anteriorly is so common beneath the weak cartilaginous portion of the ribs, and posteriorly in the neighborhood of the floating ribs. Comby mentioned the dilatation of the intercostal spaces and the paralysis of the intercostal muscles as a contributing but not an essential cause. Rummo, however, considers it of considerable importance. Light urges the importance of lessened resistance in determining the position of the pulsation, and Innocenti and Marsili regard the weakening of the chest walls as essential, and McCosh agrees with them, stating that necrosis of the ribs very commonly occurs.

7. The explanation of Wilson differs essentially from that of other investigators. He believes that "there may be (*a*) strands of atelectatic lung tissue reaching from pleural and pericardial adhesions at one extremity, by way of pleural adhesions at the other, to the surface of the chest, and thus acting as conductors of the cardiac impulse; and (*b*) the elasticity of that part of the wall of the thoracic cavity on the left side formed by the diaphragm in its relation to the fundus of the stomach is increased. The latter conditions, together with the convexity of the left wall of the heart in systolic tension, and the almost synchronous diastolic tension of the aorta of the left side of the chest, serve to explain the greater frequency of pulsation in left-sided effusion." It is conceivable that the systolic retraction might be produced by the adhesion of a sclerotic lung both to the chest wall and to the pericardium, but the results

of autopsies (the only case in favor of Wilson's theory is that of Lorenzutti) and the fact that in the great majority of cases the lung is not found adherent at the point of greatest pulsation, serve to indicate the incorrectness of this view.

The views of those who have studied the subject most carefully regarding the combination of causative factors may be briefly expressed as follows:

Toft: (1) A completely filled pleural sac; (2) dislocation of the heart; (3) involvement of the left side. Pulsation in part may be transmitted by the ascending aorta.

Traube: (1) Pulsation of the left heart; (2) weakening of the thoracic walls; (3) pericardial exudate.

Light: (1) Pressure of the empyema upon the heart; (2) vigorous heart action; (3) a certain degree of tension of the liquid; (4) an area of lessened resistance or increased tension of the mediastinum and diaphragm. Total atelectasis of the lung is not necessary.

Lépine: (1) Compressed lung; (2) diaphragm depressed and tense, but not ad maximum; (3) displacement of the heart to the right; (4) rigidity of the mediastinum.

Innocenti and Marsili: (1) Diminished resistance of the thoracic wall; (2) dislocation and violently acting heart; (3) a thickened pleura.

Béclère: (1) Left-sided effusion; (2) thickening and greater resistance of the mediastinum; (3) lessened resistance of the chest wall.

Comby: Excludes (1) alteration of the heart and bloodvessels; (2) pericarditis; (3) any peculiar character of the liquid. The chief cause is a collapsed lung. Dilatation of the intercostal spaces and muscles favors pulsation.

Fuchs: (1) Energetic heart action; (2) diminished resistance of the wall.

Auerbach: (1) The tension of the mediastinum must be greater than that of the chest wall; (2) the position of the heart toward the left favors pulsation of the left side.

Henry: (1) Left-sided large effusion; (2) relaxation of the thoracic wall from paresis of the intercostal muscles; (3) forcible heart beat.

In the case of pulsating hæmothorax that I report the obliterative pericarditis favored the increased tension of the mediastinum. The large portion of the chest wall with which the fluid was in contact served to increase the amplitude of the pulsation. With each systole of the heart it is evident that a certain amount of fluid was pumped into the chest, increasing also the amplitude of the pulsation, and that at each diastole of the heart the tension in the pleural cavity was sufficient to return a portion to the aneurysmal sac.

Pulsation appears therefore to have been due partly to the fact that the mediastinal tension was as great as that of the thoracic wall, partly to the simple pumping of fluid into the pleural cavity. Of course, the latter factor could only occur in case of ruptured aneurysm causing a hæmothorax; that in some respects would be considered a false aneurysmal sac. With the majority of those who have studied this subject, I believe that several factors are involved, but also that some of these factors are of more importance than others. The character of the fluid being of no significance, the cause must be sought in the containing walls. If there was no resistance to the continued increase in the quantity of fluid it would soon occupy the entire thoracic cavity. This resistance is found in the mediastinal tissues, which can be stretched only to a limited extent, and this stretching may be further diminished by thickening of the pleura and by pericardial and extrapericardial adhesions. The side of the mediastinum farthest away from the collection of fluid will, as a result of its greater convexity, be tensest, and, therefore, will resist the pulsation of the heart in that direction. Either as a result of depression until it becomes convex downward, when muscular contraction would tend to keep it rigid, or as a result of adhesions in the lower portion of the pleural cavity, the diaphragm also ceases to yield, and the quantity of fluid tends to remain constant. At this stage the resistance of the mediastinum, the diaphragm, and the thoracic walls must, of course, be equal, according to the physical laws of fluid under pressure in confined spaces. If then, pulsation is communicated to this fluid at any one point it will be manifested probably most distinctly at the point diametrically opposite in the containing wall. This pulsation, however, may be modified in a variety of ways. Thus, if the wall at this point is rigid and in some adjacent place is relaxed and weakened, it will be most pronounced in the relaxed portion, and it may occur that, as a result of the local yielding at this point, it will be cumulative. As long as a partly inflated lung is found in the pleural cavity, whose surface is not rendered rigid by thickened pleuræ, it will form a point of lessened resistance and absorb part or all of the pulsation. Therefore, the conditions necessary for the production of the pulsating pleurisy are: first, an accumulation of fluid under such a degree of positive pressure that the pressure upon the wall of the thorax, the mediastinum, and the diaphragm is equalized. Second, the lung must be solid or surrounded by a dense fibrous capsule, or walled off partly by the thickened pleura, partly by adhesions to the chest. Third, the existence of some local point of weakened resistance such as is produced by the empyema necessitatis, or of such a degree of elasticity of the entire thoracic wall that it pulsates as a whole. It is obvious that a thickened parietal pleura which would serve to enhance the other factors might so diminish this that pulsation would not occur.

Two other forms of pulsation occurring in the thoracic walls are of historical interest. In 1837 Stokes[1] reported a case of pulmonary carcinoma in a man, aged forty-five years, in which there was diastolic double pulsation in the upper sternal and subclavicular regions, accompanied by a bellows murmur. At the autopsy an encephaloid carcinoma of the left upper lobe was found. Graves,[2] in a case of croupous pneumonia of the right side, observed pulsation of the whole of the anterior wall of the thorax quite as vigorous as in the neighborhood of the heart. In this case, also, a bellows murmur was heard over the heart. As far as I have been able to ascertain no cases of either condition have ever been reported subsequently. Both Comby and West speak of them as practically unknown in France and England at the present time. The ability of Stokes and Graves is too great for us to question their powers of observation or diagnosis; but there can be no doubt that such cases are so excessively rare that they need hardly be considered.

The subject may be summarized as follows: Pulsation of the wall of the thorax may be due to disease of the heart or bloodvessels; may be idiopathic, that is, not the result of any definite cause; may be due to pleural or pericardial adhesions; to accumulation of fluid in the pleural cavity, or to abscess of the wall of the thorax. When the pulsation occurs as a result of accumulation of fluid in the pleura the left side is affected in the majority of cases. The patients are usually males and less than forty years of age. The mechanism depends in all probability upon the existence of a positive pressure in the pleural cavity with tension of the mediastinum.

PYOPERICARDIUM AND COMMENCING INTERSTITIAL PNEUMONIA COMPLICATING ILEOCOLITIS IN AN INFANT AGED FOUR AND A HALF MONTHS.

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PURULENT pericarditis is always a secondary affection in infancy and early childhood, and is prevalent in inverse proportion to the age of the patient. Thus of 66 cases of pericarditis seen by Baginsky under fourteen years of age, 20 were purulent, and of these 11 were in the first year of life, the oldest being seven years. Monti saw, among 35 pericarditides in children, 5 in sucklings, all of which

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[1] A Treatise on the Diagnosis and Treatment of the Diseases of the Chest, London, 1882, Part I., p. 391.

[2] Ibid., p. 292.